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## HEAT RELATED ILLNESSES

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### KEY POINTS

- Heat illnesses range in severity from mild (heat rash, heat syncope, cramps) to serious (heat exhaustion, heat injury, heat stroke).
- Although heat illness can occur in anyone, an increased risk is associated with a variety of environmental factors, personal characteristics, health conditions, and medications.
- The risk of serious heat illness can be markedly reduced by implementing a variety of countermeasures, including becoming acclimated to the heat, managing heat stress exposure, and maintaining hydration.
- Athletes, coaches, training staff, and medical personnel should be vigilant for signs and symptoms of heat related illnesses. If warning signs are acted upon and body cooling rapidly administered, serious heat illness can be avoided.
- If heat stroke is suspected, rapid body cooling by immersion or soaking in cold water or ice water should be initiated.

### INTRODUCTION

Heat stroke tragically takes the lives of apparently healthy athletes (Bergeron et al., 2005), military personnel (Carter et al., 2005), and industrial workers. From 1995 to 2001, 21 young football players died from heat stroke in the United States (Bergeron et al., 2005) and such tragic deaths continue. Several highly publicized fatalities in organized sports continue to raise awareness of heat stroke among athletes at all levels (Bailes et al., 2002; Casa et al., 2005; Roberts, 2004). Furthermore, although in recent years the incidence rate of total heat-related hospitalizations has declined in the US Army population, the incidence rate of heat stroke hospitalizations has increased five-fold (Carter et al., 2005).

Heat stroke is a persistent cause of morbidity and mortality among elderly and frail populations. On average, roughly 250 to 400 persons die from heat stroke in the United States each year, but more than 1700 persons died during the heat wave of the summer of 1980, and 700 heat-stroke deaths were documented in Chicago in 1995 (Centers for Disease Control and Prevention, 2003). A recent report indicates that from 1979 to 2002, 4780 deaths were attributable to extreme heat exposure (Centers for Disease Control and Prevention, 2005). Clearly, heat stroke is a serious public health problem.

Heat stroke is more likely to occur during exposure to hot and/or humid environments, but it can also occur in temperate environments, particularly in those performing intense prolonged physical activity. Athletes and military populations routinely perform strenuous physical activity (thus producing high levels of metabolic heat) for extended durations in hot weather and are inherently susceptible to heat stroke (Epstein et al., 1999).

The purposes of this paper are to define heat illnesses, describe their development and pathophysiology, and outline the scientific basis for their management and treatment.

## RESEARCH REVIEW

### Heat Illness Defined

**Minor heat illnesses.** Minor heat-related illnesses include heat cramps and heat syncope. Heat cramps are characterized by intense muscle spasms, typically in the legs, arms, and abdomen. Heat cramps result from fluid and sodium deficits and occur most often in persons who have not been fully acclimated to a combination of intense muscular activity and environmental heat.

**Heat syncope** (fainting) is characterized by vertigo (dizziness) and weakness during or after prolonged standing or upon rapidly standing from a lying or sitting position during heat exposure. Heat syncope results from pooling of blood in the venous circulation of the skin and of the leg muscles and occurs most commonly in dehydrated and inactive persons who are unacclimated (Seto et al., 2005).

**Serious heat illnesses.** Serious heat illnesses include heat exhaustion, heat injury, and heat stroke. These illnesses have many overlapping diagnostic features, and it has been suggested that they range on a continuum on the severity scale (Bouchama & Knochel, 2002).

*Heat exhaustion* is a mild-to-moderate illness characterized by an inability to sustain cardiac output and moderate ( $>38.5^{\circ}\text{C}$ ,  $101^{\circ}\text{F}$ ) to high ( $>40^{\circ}\text{C}$ ,  $104^{\circ}\text{F}$ ) body temperatures. It is frequently accompanied by hot skin and dehydration.

*Heat injury* is a moderate-to-severe illness characterized by injury to an organ (e.g., liver, kidney, gut, muscle) and usually, but not always, with high body temperatures  $>40^{\circ}\text{C}$  ( $104^{\circ}\text{F}$ ).

*Heat stroke* is a severe illness characterized by central nervous system dysfunction (e.g., confusion, disorientation, impaired judgment) and is usually accompanied by a core body temperature higher than  $40.5^{\circ}\text{C}$  ( $105^{\circ}\text{F}$ ). It should be noted that patients with core temperatures  $>40^{\circ}\text{C}$  do not necessarily have a heat injury or heat stroke; the entire clinical picture, including mental status and laboratory results, must be considered together. Heat stroke victims sometimes have profound impairments in brain function, characterized by cognitive changes that become apparent early. In addition, heat stroke can be complicated by liver damage, rhabdomyolysis (breakdown of muscle tissue), widely distributed blood clotting (disseminated intravascular coagulation), water and electrolyte imbalances, and kidney failure.

Heat stroke is often categorized as either “classic” or “exertional,” with the former primarily observed in the elderly or otherwise sick or compromised populations and the latter in apparently healthy and physically fit persons. There is some animal evidence that for a given heat exposure, exertional heat stroke has greater morbidity and mortality than passive (classical) heat stroke (Hubbard et al., 1997). This paper will focus primarily on exertional heat illnesses.

### Epidemiology and Risk Factors

Several personal characteristics, health conditions, medications, and environmental factors are associated with serious heat illnesses (Table 1). Although extremely rare, even those in low-risk populations (fit and heat acclimated) who take appropriate precautions and are exposed to conditions they have been exposed to many times before have incurred serious heat illness (Gardner et al., 1996; Kark et al., 1996). This suggests that some victims were inherently more vulnerable on a particular day and/or that some unique event triggered the serious heat illness.

**Table 1.** *Factors predisposing to serious heat illness*

Individual Factors	Health Conditions	Medications	Environmental Factors
Lack of Heat Acclimation	Inflammation & Fever	Anticholinergics properties (Atropine)	High Temperature
Low Physical Fitness	Cardiovascular Disease	Antiepileptic (Topiramate)	High Humidity
Excessive Body Weight	Diabetes Mellitus	Antihistamines	Little Air Motion
Dehydration	Gastroenteritis	Glutethimide (Doriden®)	Lack of Shade
Advanced Age	Skin Rash, Sunburn and Prior Burns to Large Areas of Skin	Phenothiazines (a class of antipsychotic drugs, including Thorazine®, Stelazine® and Trilafon®)	Heat Wave
	Malignant Hyperthermia	Tricyclic antidepressants (e.g., imipramine, amitriptyline)	Physical Exercise
	Sickle Cell Trait	Amphetamines, cocaine, “Ecstasy”	Heavy Clothing
		Ergogenic stimulants, e.g., ephedrine/ephedra	
		Diuretics	
		Beta-blockers (e.g., propranolol and atenolol)	

Historically, such unexpected cases were attributed to dehydration (which impairs thermoregulation and increases cardiovascular strain), but it is now suspected that a previous event (e.g., sickness or injury) might make victims more susceptible to serious heat illness (Kark et al., 1996). One theory is that previous heat injury or illness might prime the acute-phase response (i.e., increase in C-reactive proteins in the blood, fever, and other metabolic changes in response to inflammation) and augment the hyperthermia of exercise, thus inducing unexpected serious heat illness (Bouchama et al., 1993). Another theory is that prior infection might produce pro-inflammatory cytokines that deactivate the ability of cells to protect against extremely high temperatures (Sonna et al., 2004). Dehydration is associated with only about 18% of military heat stroke cases (Carter et al., 2005).

Most cases of exertional heat illness occur in the summer months (Kark et al., 1996), but the incidence rates are greatest when the weather is hottest, summer or not (Sparling, 1997). Risk for exertional heat illness in military training increases progressively as wet-bulb-globe temperatures (WBGT) rise above  $65^{\circ}\text{F}$  ( $18.3^{\circ}\text{C}$ ); cases occur primarily with strenuous exercise (e.g., running) and after several consecutive days of hot weather (Kark et al., 1996). A lack of heat acclimation is an important factor for heat intolerance (extreme sensitivity to the heat or discomfort in the heat) and heat illness (Armstrong et al., 1990a). Likewise, US Army recruits from northern states are more susceptible to heat illness than are recruits from southern states (Carter et al., 2005). This latter observation suggests that persistent heat exposure early in life might provide protective benefit later.

There is evidence that the exertional heat stroke cases are increasing. The US Army has witnessed a dramatic increase in heat stroke hospitalizations over the last decade, despite reductions in less serious heat illnesses (Carter et al., 2005). The reasons for this increase in heat stroke hospitalizations are unclear but may include an increased emphasis on running activities in military training (Departments of Army and Air Force, 2003) and possibly an increased use of ephedra-containing nutritional supplements (Oh & Henning, 2003). Heat stroke fatalities associated with American football declined significantly from 1960 to 1990, but since 1994, they appear to be increasing again; this rise may be associated, in part, with increased use of certain nutritional supplements (Bailes et al., 2002). The use of stimulants (e.g., ephedra, cocaine, heroin, and methamphetamine) is associated with an increased risk of heat stroke (Crandall et al., 2002; Marzuk et al., 1998). Stimulants increase metabolic heat production and may impair heat dissipation, thus elevating body temperature (Crandall et al., 2002). For example, epidemiological data gathered from three large US metropolitan areas have also demonstrated that the risk of death from cocaine overdose increases sharply during hot weather (Marzuk et al., 1998).

A high body mass index (BMI) and poor fitness are also important risk factors for heat-related illnesses. Marine Corps recruits increase their risk for exertional heat illness by three-fold with either a BMI of  $\geq 22 \text{ kg/m}^2$  or a time for running 1.5-miles  $\geq 12$  min. Furthermore, for those recruits having both large body masses and slow running times, the heat-illness risk increased eight-fold (Gardner et al., 1996). Although high BMI and slow initial run time were important predictors for exertional heat illness during the first week of basic training, by week 14 (final week), only slow run time was predictive of exertional heat illness (Wallace et al., 2006).

There are race (ethnicity) and gender influences on the risk for exertional heat illness. Blacks and Hispanics are less likely than Caucasians to be hospitalized for exertional heat illness. Likewise, Caucasian women are hospitalized for exertional heat illness about four times more often than their Black and Hispanic counterparts (Carter et al., 2005).

Genetic disorders can modify the risk of exertional heat illness. Case reports suggest that sickle cell trait (possessing one of two genes required to cause abnormal hemoglobin production and distortion or "sickling" of red blood cells) may increase the risk of serious heat illness (Kerle & Nishimura, 1996). Sickle cell trait is more prevalent in Blacks and certain Asian populations, and these populations are at increased risk if they are also in poor physical condition, dehydrated, and exposed to hot environments. Erythrocyte sickling can reduce blood flow and oxygen carrying capacity of red blood cells and lead to damage of the capillary walls, blood coagulation, and localized damage to tissues due to inadequate blood supply. Also, persons susceptible to malignant hyperthermia, a genetic disease characterized by extreme muscle contractions and a rapid rise in body temperature when exposed to certain anesthetics, may be at greater risk for exertional heat illness (Muldoon et al., 2004).

## Heat Stress Responses and Development of Heat Illness

Heat stress refers to environmental and metabolic processes that increase body temperature. Metabolic heat is released from active skeletal muscles and transferred from the body core to the skin, where it is dissipated primarily by evaporation of sweat. If this heat is not dissipated, core temperature will rapidly increase. Heat exchange from the skin to the environment is impaired by hot air temperatures, high humidity, poor movement of air around the skin, solar radiation, radiation from hot surfaces (e.g., boulders, soil, buildings), and clothing, including protective athletic gear such as shoulder pads and helmets (Brothers et al., 2004) used in American football (Bergeron et al., 2005; Fowkes et al., 2004; Kulka & Kenney, 2002).

Body temperature also increases during febrile illness. Fever results in an elevated thermoregulatory set-point, the body temperature at which sweating and blood flow to the skin increase to dissipate heat. Persons with fevers have accentuated rises in body temperature during heat exposure and physical exercise, and these rises may involve increases in prostaglandins and other inflammatory mediators such as cytokines (Leon, 2006).

Prior injury or infection may provoke exertional heat illness (Sonna et al., 2002). Figure 1 depicts a laboratory experiment for a subject subsequently shown to have had an infected blister (cellulitis). This individual demonstrated an unusually rapid rise in body temperature when performing physical exercise in the heat.

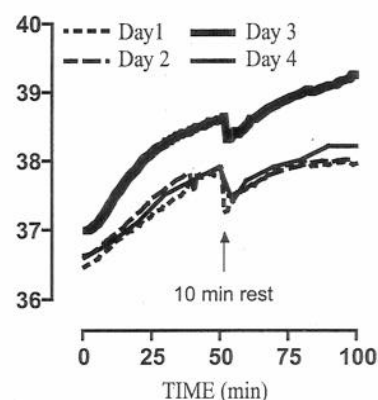


FIGURE 1. Prior injury or infection may provoke exertional heat illness.

The figure represents a laboratory experiment for a subject who had normal core temperature responses to a standard exercise bout in the heat on days 1 and 2. On day 3, he exhibited "abnormal" core temperature responses to the exercise session (depicted as the bold solid line). After completing the exercise bout, the subject complained of an infected blister, which required immediate medical attention. After receiving two days of oral antibiotic treatment for the bacterial infection, core temperature responses to exercise returned to normal values (Day 4). (USARIEM, unpublished).

Environmental and exercise heat stress challenge the cardiovascular system to provide a rapid flow of blood to the skin, where it tends to pool, reducing the return of blood to the heart. To compensate, blood flow is diverted from the liver, kidneys, and intestines to better supply muscle, skin, brain, heart, and lungs. This reduced blood flow to the viscera can cause insufficient oxygenation of those tissues, release of bacterial toxins into the blood, tissue damage caused by the generation of oxidants and nitric oxide, and excessively high tissue temperatures or heat shock ( $>41^{\circ}\text{C}$ ,  $105.8^{\circ}\text{F}$ ) (Lambert, 2004).



Hyperthermia (excessively high body temperature) has a direct effect at the cellular level, with the magnitude and duration of heat shock influencing whether cells respond by adaptation known as Acquired Thermal Tolerance (ATT) (Kregel, 2002), tissue injury, or cell death (Gabai & Sherman, 2002). Hyperthermia, insufficient tissue blood supply, and the systemic inflammatory response can result in cellular dysfunction, loss of cell membrane integrity, disseminated intravascular coagulation (blood clotting throughout the body), and dysfunction of multiple organ systems, especially the lungs, liver and kidneys. At the molecular level, hyperthermia can lead to protein degradation. Altered metabolic pathways due to hyperthermia can accelerate chemical reaction rates and contribute to apoptosis – programmed cell death. Central nervous system dysfunction is mediated by reduced cerebral blood flow, heat shock influences on neural tissue metabolism, and disordered blood coagulation.

### Adaptation to Heat Stress

Heat acclimation develops over a period of days and weeks through repeated heat exposures that are sufficiently stressful to elevate both core and skin temperatures and to provoke profuse sweating. Heat acclimatization refers to adaptations that occur over months and years of exposure to a hot climate. In this paper, the focus is on heat acclimation. Heat acclimation induces biological adjustments that reduce the adverse physiologic effects of heat stress and improve exercise performance during hot weather exposure. Aerobic exercise training in temperate climates also has beneficial effects on physiology and exercise performance in the heat, but exercise training alone cannot replace the benefits of heat acclimation.

Table 2 provides a brief description of the benefits of heat acclimation, which minimizes heat strain, i.e., the physiological adjustments to a given heat exposure, and improves submaximal aerobic exercise capability. These benefits of heat acclimation are achieved by expansion of plasma volume, improved sweating and skin blood flow responses, and better fluid balance and cardiovascular stability. Heat acclimation is specific to the climate (hot dry versus hot humid) and physical activity level, i.e., acclimation to the hot, dry heat is better for adapting an individual to exercise in the dry environment than is acclimation to hot, humid heat, and vice versa. However, heat acclimation to humid or dry climates can markedly improve exercise capabilities in the other hot climate.

**Table 2** *Benefits of heat acclimation*

Heat Strain – Minimized	Exercise Performance – Improved
<p><i>Core Temperature – Reduced</i></p> <ul style="list-style-type: none"> <li>▪ <b>Sweating – Improved</b> <ul style="list-style-type: none"> <li>- Earlier onset of sweating</li> <li>- Higher rate of sweating</li> <li>- Improved distribution of sweat on skin*</li> <li>- Reduced failure of localized sweating*</li> </ul> </li> <li>▪ <b>Skin Blood Flow – Improved</b> <ul style="list-style-type: none"> <li>- Earlier onset</li> <li>- Higher rate*</li> </ul> </li> <li>▪ <b>Metabolic Rate – Lowered</b></li> </ul>	<p><i>Cardiovascular Stability – Improved</i></p> <ul style="list-style-type: none"> <li>▪ <b>Heart rate – Lowered</b></li> <li>▪ <b>Stroke volume – Increased</b></li> <li>▪ <b>Blood pressure – Better Defended</b></li> <li>▪ <b>Myocardial compliance – Improved</b></li> <li>▪ <b>Fluid balance – Improved</b></li> <li>▪ <b>Thirst – Improved</b></li> <li>▪ <b>Sodium loss – Reduced (sweat &amp; urine)</b></li> <li>▪ <b>Total body water – Increased</b></li> <li>▪ <b>Blood &amp; plasma volume – Increased &amp; Better Defended</b></li> <li>▪ <b>Acquired Thermal Tolerance – Improved</b></li> </ul>

\* When acclimated to a hot, humid environment

Acquired Thermal Tolerance refers to cellular changes from a severe non-lethal heat exposure that allows the organism to survive a subsequent and otherwise lethal heat exposure. Acquired Thermal Tolerance and heat acclimation are complementary; acclimation reduces the adverse effects of heat on physiology, whereas Acquired Thermal Tolerance increases survivability to a given heat load (Kregel, 2002). Acquired Thermal Tolerance is associated with the production of specialized proteins (heat shock proteins) that bind to various molecules to provide cellular protection and to accelerate tissue repair. In addition to the actions of heat shock proteins, other pathways and cellular systems likely contribute to Acquired Thermal Tolerance (Sonna et al., 2002).

**Table 3** *Heat acclimation strategies*

<p><b>Must mimic the climate of athletic event or occupational setting</b></p> <p><b>Include adequate heat stress</b></p> <ul style="list-style-type: none"> <li>▪ Heat must be sufficient to cause heavy sweating</li> <li>▪ Use exercise/rest cycles to intensify or diminish the effect of the heat stress on bodily functions</li> <li>▪ Include at least 6-14 days of adequate heat stress</li> <li>▪ Exercise daily for at least 90 minutes</li> </ul> <p><b>Start with acclimation and exercise training</b></p> <ul style="list-style-type: none"> <li>▪ Be flexible in scheduling training</li> <li>▪ Build confidence</li> <li>▪ Performance benefits may take longer to achieve than physiological benefits</li> </ul> <p><b>Methods of heat acclimation</b></p> <ul style="list-style-type: none"> <li>▪ Climate-controlled room (sauna or heat chamber) or hot weather</li> <li>▪ Incorporate training by including additional acclimation sessions</li> </ul> <p><b>Days leading to athletic performance or event</b></p> <ul style="list-style-type: none"> <li>▪ Start slowly and decrease training duration and intensity, limit heat exposure</li> <li>▪ Acclimatize in heat of the day</li> <li>▪ Train in coolest part of the day</li> <li>▪ Use appropriate work/rest cycles</li> <li>▪ Be vigilant of salt and fluid needs, especially during the first week of acclimation</li> </ul>
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Table 3 provides heat acclimation strategies that can be considered for preparation for athletic events or occupational tasks in hot weather. The primary benefits to heat acclimation are increased physical work output associated with improved thermoregulatory and cardiovascular function and reductions in the risk of experiencing heat-related illness. A minimum heat exposure of 90 minutes per day for 8-14 days with some physical exercise in the heat is required for appropriate adaptation (Sawka et al., 1996). Longer heat exposures and more extended acclimation will likely provide additional benefits (Sawka et al., 2001). Persons with prior heat illness might require more time to become acclimated (Armstrong et al., 1990b).

### Prevention and Immediate Treatment of Heat Illness

Most serious heat illness cases can be prevented by inducing heat acclimation and acquired thermal tolerance, by avoiding exposure to overwhelming heat stress, and by maintaining adequate hydration. In addition, coaches and trainers should be aware of the signs and symptoms of heat serious heat illness (Table 4). Athletes displaying these signs and symptoms should discontinue exercise and be allowed to recover in cool a environment with replenishment of lost body fluids and electrolytes.

**Table 4** Warning signs and symptoms of exertional heat illnesses (modified from Binkley et al. (2002) and Department of the Army and Air Force (2003).

Signs of Possible Heat Exhaustion	Signs of Possible Heat Stroke
<ul style="list-style-type: none"> <li>▪ Rapid pulse</li> <li>▪ Rectal temperature usually &lt;104°F (40.0°C)</li> <li>▪ Headache</li> <li>▪ Dizziness/fainting</li> <li>▪ Nausea/vomiting</li> <li>▪ Unsteady walk</li> <li>▪ Weakness</li> <li>▪ Muscle cramps</li> <li>▪ Chills/Goosebumps</li> </ul>	<ul style="list-style-type: none"> <li>▪ Weak and rapid pulse</li> <li>▪ Rectal temperature usually &gt;105°F (40.5°C)</li> <li>▪ Central nervous system changes <ul style="list-style-type: none"> <li>– Confusion/disorientation</li> <li>– Agitation/aggressiveness</li> <li>– Blank stare/apathy</li> <li>– Irrational behavior</li> <li>– Staggering gait</li> <li>– Delirium</li> <li>– Convulsions</li> <li>– Unresponsiveness, coma</li> </ul> </li> <li>▪ Hot and wet or dry skin</li> <li>▪ Vomiting</li> <li>▪ Involuntary bowel movement</li> <li>▪ Hyperventilation</li> </ul>

When preventive measures fail, the management of serious heat illness includes external cooling of the body, monitoring the victim, and possibly rehydration. Body cooling lowers tissue temperatures, stabilizes blood pressure by constricting blood vessels in the skin, and is an important factor in positive recovery and prognosis (Heled & Deuster, 2006). Immersion of the body or soaking the skin in cool or iced water with skin massage are the most effective methods, but using cool showers and ice-cold sheets and ice packs, along with removing unnecessary clothing are effective in dissipating the body heat load (Casa et al., 2005). Ice water immersion reduces body core temperature by 0.15°-0.30°C per minute (Casa et al., 2005; Costrini, 1990). Although less effective than ice water, using fans with cool water reduces core body temperature at a rate of 0.06°C per minute (Casa et al., 2005). Body cooling should be immediately initiated with the most effective method available and continued until rectal temperature falls below 38.5°C (101.8°F) (Proulx, 2003). Fluid and electrolyte deficits should be corrected; this can be achieved by drinking or by intravenous administration of appropriate fluids.

## SUMMARY

Persons participating in strenuous physical activity for extended durations in hot weather are susceptible to exertional heat illness. Exertional heat illnesses range from mild to serious medical situations. Many factors—environmental, physical, physiological, medical, and drug-related—are associated with an increased risk of serious heat illnesses. Athletes, team personnel, and medical staff can mitigate risks by identifying high-risk persons, by following guidelines for heat acclimation, fluid and electrolyte replacement, and exercise/rest strategies, and by vigilance. When serious heat illness occurs, body cooling should start immediately. Immersion and soaking in cooled or iced water is the most effective method of rapidly cooling the body.

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**S U P P L E M E N T**

**AVOIDING HEAT ILLNESS BY BEING S.M.A.R.T.**

Protecting your health during physical activity in hot climates is as simple as remembering to keep cool and using common sense. Exposure to heat can lead to serious, even life-threatening, illness, as witnessed by the deaths of elite athletes during sports training and competition and by the deaths of hundreds of persons during heat waves around the world. Heat illness is preventable in most cases! Follow the basic tips below and avoid heat injuries by being S.M.A.R.T.

**Schedule** workouts carefully to avoid the highest temperatures. Limit your workouts to morning and evening hours to avoid peak summer heat.

**Monitor** high risk athletes. Coaches, athletes, athletic trainers, and athletes should learn the signs and symptoms of serious heat-related illnesses and be on the lookout for those who might be at risk. Athletes who have been recently ill, for example, with the flu or a muscle injury, who have a prior history of heat illness, who are unacclimated to exercise in the heat, and who are highly motivated to succeed in their sports are at the greatest risk.

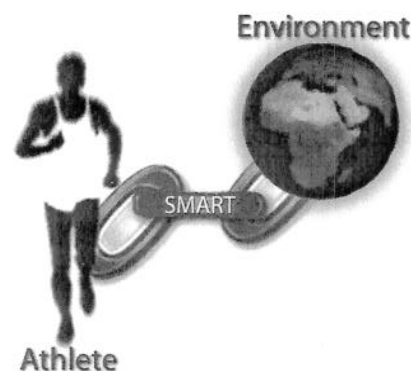
*Warning signs and symptoms of exertional heat illnesses* (modified from Binkley et al. (2002) and Department of the Army and Air Force (2003)).

**Signs of Possible Heat Exhaustion**

- Rapid pulse
- Rectal temperature usually <104°F (40.0°C)
- Headache
- Dizziness/fainting
- Nausea/vomiting
- Unsteady walk
- Weakness
- Muscle cramps
- Chills/Goosebumps

**Signs of Possible Heat Stroke**

- Weak and rapid pulse
- Rectal temperature usually >105°F (40.5°C)
- Central nervous system changes
  - Confusion/disorientation
  - Agitation/aggressiveness
  - Blank stare/apathy
  - Irrational behavior
  - Staggering gait
  - Delirium
  - Convulsions
  - Unresponsiveness, coma
- Hot and wet or dry skin
- Vomiting
- Involuntary bowel movement
- Hyperventilation





**Acclimate and Adjust** workouts and clothing to the environment. Become acclimated to the heat for at least 8-14 days by gradually increasing exercise time and intensity in the heat. Reduce work intensity and time of heat exposure in extremely hot, humid conditions. Wear light-colored and loose-fitting clothing.

**Recover and Replenish.** Recover in cool conditions for a long enough time to reduce body temperature, and remember to drink during exercise to replenish fluids and electrolytes. Sports beverages can help replace the fluids and minerals that you lose through sweat.

Think about emergency procedures to cool athletes who may suffer heat illness. Have plans ready to rapidly administer effective body cooling procedures, especially by immersing the athlete in cold water or ice water.

### SUGGESTED ADDITIONAL RESOURCES

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